

coagulation is described in outline, but adequately for an understanding of the physiologic basis of the more commonly used laboratory tests. The specific diagnoses of blood coagulation diseases are discussed with great brevity, as are the principles of treatment. For example, less than a paragraph is devoted to von Willebrand's disease, although this is one of the commonest of all the hereditary blood diseases. It might be added that while the author is correct when he states that the diagnosis of this disorder is established with certainty when the triad of prolonged bleeding time, depressed platelet adhesiveness and low factor VIII is found, most cases do not manifest this triad; moreover, in the last two or three years an immunologic test which should be performed in every patient in whom this disorder is suspected

has been introduced, and this test is not mentioned at all in this text. The treatment of specific bleeding episodes such as hemarthroses is not discussed. Accordingly, the book cannot be recommended for the medical practitioner who has the primary responsibility of diagnosis and management of a bleeding disorder. However, for others, especially medical students, the book can be recommended as a very readable, exceptionally well-written account, containing all the basic information. It is one of the best and shortest accounts of blood coagulation that I have read.

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Literature Briefs

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Literature briefs were submitted by Drs. I. C. Andrews, R. Clark, A. Goldblatt, J. Levitt, and W. Mannheimer. Briefs appearing elsewhere in this issue are part of this column.

Circulation

HEMODYNAMICS OF HEAT STROKE

Two types of circulatory responses were found in eight patients studied following heat stroke. Seven of the eight had high cardiac indices (6.45 l/min/m^2) and low systemic vascular resistance ($603 \text{ dynes/sec}^2/\text{cm}^2$), while one patient had a hypodynamic response with a low CI (1.7 l/min/m^2) and elevated CVP, $18 \text{ mm H}_2\text{O}$, and a mean arterial pressure of 36 mm Hg . Mean rectal temperature was 41.5°C in the hyperdynamic group and 43.3°C in the lone hypodynamic patient. The results obtained demonstrate that survival depends upon an adequate cardiovascular response. For a time it must be capable of greater than normal cardiac output to meet the elevated demand. The two principal mechanisms involved are cutaneous vasodilatation to dissipate heat and reduced TPVR despite normothermia, probably associated with tissue injury. The data in this study demonstrated that the circulatory failure in

heat stroke was primarily right-sided heart failure, but it did not define whether it was due to myocardial failure or to elevated pulmonary vascular resistance. Prompt therapy consisting of rapid cooling, modest fluid replacement, and myocardial stimulation when needed resulted in no death in 36 cases of heat stroke. (O'Donnell, T.F., and Clowes, G.H.A.: *The Circulatory Abnormalities of Heat Stroke*. *N. Engl J Med* 287: 734-737, 1972.)

ANEMIA OF CHRONIC DISEASE In 58 patients with malignancy, rheumatoid arthritis, chronic infection, or other chronic debilitating disease, hematocrits correlated well with serum albumin and transferrin values and poorly with serum iron, vitamin B_{12} , and gamma-globulin levels. There was no correlation between marrow iron stores and serum iron-binding capacity. "Simple chronic anemia" appears to result from impaired synthesis of all proteins, with resultant decrease in the proteins involved in hematopoiesis. (Kurnick, J.E., and others: *Mechanism of the Anemia of Chronic Disorders*. *Arch Intern Med* 130: 323, 1972.)